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The n-3 polyunsaturated fatty acid status in the Hangzhou regionD Li¹, XM Yu², YH Zhang¹, T Yao¹, XQ Zhou², AJ Sinclair³¹Department of Food Science and Nutrition, Zhejiang University, Hangzhou, China, 310029²Clinical laboratory, Zhejiang Hospital, Hangzhou, China³Department of Food Science, RMIT University, Melbourne, VIC, 3000

Background – Increased dietary intake of n-3 polyunsaturated fatty acid (PUFA) raises n-3 PUFA levels in tissues, and is associated with beneficial effects on the prevention of cardiovascular diseases and inflammation, and perhaps with neuropsychiatric disorders.

Objective – To investigate the n-3 PUFA status in the Hangzhou region in China by determination of the serum phospholipid (PL) fatty acid composition, as a biomarker of status.

Design – Cross-sectional study of 154 free-living subjects (108 males and 46 females) recruited from Hangzhou, China. Each subject gave a fasting blood sample, serum phospholipid was separated by thin liquid chromatography. Fatty acid methyl esters were prepared by standard methods, and separated by gas liquid chromatography.

Outcomes – The ages were 55.9 ± 13.7 and 55.6 ± 10.1 yrs, and BMI were 23.9 ± 3.1 and 22.6 ± 3.1 kg/m² for males and females, respectively. Table shows the serum PL composition of total and individual n-3 PUFA for both genders (as percent of PL fatty acids).

	Male (n=108)	Female (n=46)
18:3n-3	0.7 ± 0.2	0.8 ± 0.3
20:5n-3	2.1 ± 0.8	2.2 ± 1.0
22:5n-3	0.6 ± 0.3	0.5 ± 0.3
22:6n-3	5.3 ± 2.0	5.7 ± 2.2
Total n-3 PUFA	8.6 ± 2.0	9.2 ± 2.0

Conclusions – Compared with our previous study from Australian populations, where the total n-3 PUFA was found to be 5.9% of PL fatty acids¹, the higher proportion of 20:5n-3, 22:6n-3 and total n-3 PUFA in serum PL reported here may contribute to the lower coronary heart disease incidence in the Hangzhou population.

1. Li D, Sinclair AJ, Mann N, Turner A, Ball M, Kelly F, Abedin L, Wilson A. The association of diet and thrombotic risk factors in healthy male vegetarians and meat-eaters. *Eur J Clin Nutr* 1999;53:612-19.

Effects of exposure to grape-seed polyphenols and vitamin C on lipid peroxidation in vivo

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Introduction - Oxidative stress has been implicated in a number of disease processes. There is evidence suggesting that vitamin C, a major water-soluble antioxidant, may reduce oxidative stress. The effects of dietary polyphenols, water-soluble compounds with potent antioxidant activity *in vitro*, on oxidative stress are unclear.

Objectives - The objectives of this study were to investigate the effect of supplementation with grape-seed polyphenols on oxidative stress, and to compare any effects to those of vitamin C.

Design - Following a 3-week washout, participants were randomised to receive (i) 500mg/day vitamin C + matched placebo (n = 19), (ii) 1000mg/day polyphenols + matched placebo (n = 16), (iii) 500mg/day vitamin C + 1000mg/day polyphenols (n = 16), or (iv) matched placebos (n = 18). Plasma and urinary F₂-isoprostanes and oxidised low-density lipoproteins were analysed as markers of oxidative damage.

Outcomes - Supplementation with grape-seed polyphenols resulted in a significant increase in urinary excretion of specific phenolic acids (3-hydroxyphenylpropionic acid), but did not alter F₂-isoprostane concentrations or oxidised low-density lipoproteins. The phenolic acid metabolites, markers of exposure to grape-seed polyphenols, were not related to changes in markers of oxidative stress. Plasma vitamin C levels increased significantly following supplementation. Plasma F₂-isoprostane concentrations fell following supplementation with vitamin C (p=0.056). There was no change in urinary F₂-isoprostane concentrations or oxidised low-density lipoproteins. There was no relationship between increases in plasma vitamin C and changes in markers of oxidative stress.

Conclusions - These results support the suggestion that supplementation with vitamin C may reduce *in vivo* lipid peroxidation. However, supplementation with grape-seed polyphenols and exposure to phenolic acid metabolites had no effect on *in vivo* lipid peroxidation.